SCHOLARLY REVIEW

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The epidemiology of hidradenitis suppurativa

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Summary

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease defined clinically by multiple, painful inflammatory lesions occurring predominantly in flexural sites. Onset is typically soon after puberty; however, it remains unknown whether the menopause induces remission. In North American and European patients with HS the female-to-male ratio is approximately 3 : 1 but the ratio is 1 : 2 in South Korean patients. It may be that some elements of HS epidemiology cannot be generalized across all populations. Elements of HS epidemiology in the USA and Europe are well established, including strong associations with obesity and smoking, which may increase disease severity. There are associations between HS and other cardiovascular disease (CVD) risk factors, including type 2 diabetes and metabolic syndrome. People with HS have double the risk of death from CVD compared with those without HS and 1·5 times the risk compared with patients with psoriasis. Depression and anxiety are associated with HS and completed suicide rates in those with HS are more than double the rates in controls. Associations exist between HS and other chronic inflammatory conditions, particularly inflammatory bowel disease and inflammatory arthritis. Case–control studies demonstrate associations with pilonidal sinus, polycystic ovary syndrome, Down syndrome, obstructive sleep apnoea and pyoderma gangrenosum. Population-based studies using routinely collected healthcare data from the USA estimate a prevalence of 0·1%, suggesting HS is relatively uncommon. European studies include undiagnosed patients and typically estimate prevalence of 1% or more, suggesting a common condition. Resolving the controversy surrounding a greater than 10-fold difference in HS prevalence estimates remains a high priority.

What is already known about this topic?

- Hidradenitis suppurativa (HS) prevalence figures vary more than 80-fold, from 0.05% to 4.1%.
- In North America and Europe, HS is most prevalent in adult women of working age.
- HS is linked to smoking and obesity.

What does this study add?

- Wide variation in HS prevalence estimates is probably due to differing data sources, different levels of recognition of HS and variable inclusion of undiagnosed cases.
- HS is associated with other chronic inflammatory diseases, cardiovascular disease (CVD) and depression.
- Mortality rates from CVD are more than 50% higher in HS than in psoriasis and there is an increased risk of completed suicide compared with controls.

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Hidradenitis suppurativa (HS) is a chronic skin disease characterized by painful inflammatory nodules and abscesses in flexural sites that exude pus and often lead to scarring.[1] Investigation of the epidemiology of HS is challenging because the diagnosis is often delayed or never formally established. Average diagnostic delay for HS is 7·2 years, compared with 1·6 years for psoriasis.[2]

Case definition

Determination of HS prevalence and incidence requires a standard disease definition. The definition of HS is clinical, with no diagnostic test. The original disease definition was developed by consensus at two international HS meetings in 2006 and 2009, resulting in the following description: 'a chronic, inflammatory, recurrent, debilitating, skin follicular disease that usually presents after puberty with painful deep seated, inflamed lesions in the apocrine gland-bearing areas of the body, most commonly, the axillary, inguinal and anogenital regions'.[3,4] There are three key elements to the diagnosis: (i) characteristic inflamed skin lesions; (ii) predominantly flexural sites, in particular the axillae and groin; and (iii) chronicity. The gold-standard case definition is from history-taking and in-person examination by a clinician who is experienced in HS care, including features such as characteristic scarring and skin tunnel (also known as sinus tract or skin fistula) formation, which may be present.

Prevalence

The prevalence of HS remains a highly controversial subject, with figures for Europe and North America ranging from 0·05% to 4·1%, an 82-fold difference.[5,6] How can estimates of similar populations vary so widely? The answer may relate to the diversity of methodologies and data sources utilized. Table 1 lists the different methods and provides examples of the prevalence figures obtained. The highest figure of 4·1% is based on an examination of 507 Danish young adults, mean age approximately 27 years old, undergoing screening for sexually transmitted diseases (STDs).[6] There were 20 cases of HS, in six men and 14 women. The study used the gold-standard case definition of in-person examination by a dermatologist. The relatively high prevalence figure may in part relate to the age range of participants, who were mainly in their third and fourth decades and could also have been influenced by case ascertainment bias in terms of STD clinic attendees being concerned about genital HS lesions.

The lowest HS prevalence figure used insurance data from the USA to estimate period prevalence in 2007·[5] There are several reasons why the figure is likely to be an underestimate: (i) insurance data misses uninsured patients who probably have a higher prevalence of HS because it is more common in lower socioeconomic groups;[7] (ii) only diagnosed patients and those receiving a HS-specific procedure were included, missing undiagnosed patients estimated to be at least one-third of the total;[8,9] (iii) case ascertainment relies on clinical recognition of HS, which may vary between locations and over time; and (iv) population-based data relies on accurate diagnostic coding.

Prevalence in studies from the USA tends to be lower than in European studies. Could the difference be due to variation in ethnicity and genetics between the populations of the USA and Europe? Prevalence in African-Americans was three times higher, rather than lower, than in white individuals,[10] which counts against this argument. It is more likely that the difference relates to the differing data sources and methodologies used in studies of US and European populations.

Epidemiology data for Asian populations are more limited. A population-based cross-sectional study of the South Korean National Health Insurance database using International Classification of Diseases, 10th revision codes found a 10-year period prevalence of 0·06%.[11] Prevalence in Australia, using a validated screening questionnaire, was 0·67% [95% confidence interval (CI) 0·53–0·84%].[12] Data for Africa are currently limited; however, a recent questionnaire study conducted in Ghana found a prevalence of 0·8%.[13]

Incidence

In keeping with the wide variation in prevalence estimates between studies, incidence figures from population-based studies also show wide variation. Annual incidence in the USA was 11·4 (95% CI 11·1–11·8) cases per 100 000 population,[14] while in the UK it was 28·3.[8] It has been hypothesized that HS incidence rates may be increasing due to increasing recognition of the condition. In support of this hypothesis, population-based data from the USA show that HS incidence in 2015–16 was one-third higher than the average annual incidence during the decade from 2006 to 2016·[14] In contrast, the UK study found a relatively static incidence rate.[8]

Demographics

In European and North American populations, HS has a clear female preponderance, with a female-to-male ratio of approximately 3 : 1·[8,10,15] The reverse was found in South Korea, with a female-to-male ratio of approximately 1 : 2·[11,16] Prevalence is highest in the fourth and fifth decades of life.[8,10,15] Onset of HS typically occurs in young adulthood, with a new diagnosis being most common in people aged 18–29 years of age in the USA.[14] Factoring in a typical diagnostic delay of 7 years,[2] this fits with the hypothesis that onset occurs at or soon after puberty in many patients. Reports of prepubertal HS are limited to small case series, reflecting its relative rarity.[17] Association is reported between HS and lower socioeconomic status, which may in part relate to higher prevalence of HS risk factors in this group, or could be a consequence of HS.[7]

Environmental associations

Smoking and obesity are the two environmental factors most strongly linked to HS (Table 2). A systematic review including

Table 1 Methodology, population and data sources used for hidradenitis suppurativa prevalence estimate in North America and Europe[a]

Methodology	Population	Data source	Prevalence estimate, % (95% CI)	Reference (first author, year)
Population-based, diagnostic codes	Diagnosed cases from 15 million individuals in the USA	Insurance claims	Period prevalence in 2007: 0·053 (0·051–0·054)	Cosmatos 2013[5]
Population-based, diagnostic code	Diagnosed cases from 48 million individuals in the USA	Insurance claims and self-pay records	0·10 (0·097–0·099)	Garg 2017[10]
Population-based, diagnostic codes	Diagnosed cases from 144 000 individuals in Olmsted County, USA	Rochester Epidemiology Project, centralized database for most healthcare providers	0·13 (0·11–0·15)	Shahi 2014[58]
Population-based, diagnostic code and algorithm for multiple flexural skin boils	Diagnosed and undiagnosed cases from 4·3 million individuals in the UK	Routinely collected primary care CPRD database	0·77 (0·76–0·78) Including probable cases: 1·19 (1·18–1·20)	Ingram 2018[8]
Cross-sectional study: positive responses to survey question about painful flexural boils	10 000 members of the general French population	6887 questionnaire responses in those aged 15 years or older	Period prevalence in previous 12 months: 0·97	Revuz 2008[15]
Cross-sectional study: positive responses to two survey questions about skin boils	Members of the general Danish population	16 404 questionnaire responses (49% response rate) in those aged 30 years or older	2·10 (1·88–2·32)	Vinding 2014[59]
In-person examination by dermatologists	507 patients, male-to-female ratio 1·2 : 1, mean age 27 years, undergoing screening for sexually transmitted diseases	History and examination findings	4·1 (3·0–6·0)	Jemec 1996[6]

[a]References are examples, rather than an exhaustive list. CI, confidence interval; CPRD, Clinical Practice Research Datalink.

6174 HS cases and 24 993 controls demonstrated an odds ratio (OR) of 4·34 (95% CI 2·48–7·60) for HS and current smoking and an OR of 3·45 (95% CI 2·20–5·38) for HS and obesity.[18] Both higher smoking pack-years and higher body mass index (BMI) have been linked to greater disease severity.[19] In multivariate analysis, an increase in one unit of BMI was associated with an increase of 0·84 units in mean Sartorius HS severity score.[20] It should be noted that there is a relative lack of prospective longitudinal cohort data to confirm whether smoking and obesity are true risk factors for HS initiation.

Association with other medical conditions

Follicular occlusion

A link between HS and pilonidal sinus is expected because both are characterized by follicular occlusion. Case–control studies demonstrate five-fold higher odds of pilonidal sinus in those with HS compared with controls,[8,11] one of the strongest associations between HS and other medical conditions (Table 3). It is likely that pilonidal sinus is part of the HS disease spectrum.[21]

Table 2 Environmental risk factors/associations from European and North American hidradenitis suppurativa case–control studies[a]

	Odds ratio (95% CI)		
Reference (first author, year)	Current smoker	Past smoker	Obesity[b]
Revuz 2008[15,c]	12·55 (8·58–18·38)	1·46 (0·86–2·46)	4·42 (2·82–6·93)
Shlyankevich 2014[37]			2·09 (1·03–4·22)
Ingram 2018[8]	3·61 (3·44–3·79)	1·93 (1·81–2·05)	3·29 (3·14–3·45)
Shalom 2015[60]			1·74 (1·56–1·94)
Miller 2014[61,d]			2·58 (2·00–3·23)

[a]References are examples, rather than an exhaustive list; [b]obesity is defined as a body mass index greater than 30; [c]medically assessed patients (n = 302); [d]population-level data shown (n = 326). CI, confidence interval.

Acne conglobata is strongly associated with HS in Korean patients (prevalence 4·5%; OR 5·07, 95% CI 4·69–5·49)[11] and the association remains relatively strong between HS and acne vulgaris in UK patients (OR 1·77, 95% CI 1·66–1·88).[8]

Metabolic syndrome

Metabolic syndrome is typically defined by at least three of: abdominal obesity, dyslipidaemia, hypertension and diabetes mellitus. Regarding dyslipidaemia, a systematic review of case–control studies found associations between HS and hypertriglyceridaemia (pooled OR 1·67, 95% CI 1·14–2·47) and low high density lipoprotein (OR 2·48, 95% CI 1·49–4·16).[18] Meta-analysis investigating a possible association between HS and type 2 diabetes gave a pooled OR of 2·85 (95% CI 1·34–6·08)[18] and a subsequent review of a subset of adjusted analyses gave a pooled OR of 1·69 (95% CI 1·50–1·91).[22] While several individual case–control studies have demonstrated a statistically significant association between HS and hypertension, meta-analysis of the data available in 2015 gave a nonsignificant result in the context of a relatively wide 95% CI (OR 1·5, 95% CI 0·63–3·58).[18] Nevertheless, adding in the strong association between HS and obesity, there is a 40% prevalence of metabolic syndrome in people with HS.[23] Meta-analysis of available studies confirmed an OR of 2·22 (95% CI 1·62–3·06) for metabolic syndrome in HS compared with controls.[18] Insulin resistance is also increased in HS, present in 43% of those with HS compared with 16% of controls.[24]

Cardiovascular disease

Does the higher prevalence of cardiovascular disease (CVD) risk factors including smoking and metabolic syndrome in people with HS translate into higher rates of CVD and CVD-related mortality? The answer was provided in 2016 by a population-based cohort study using Danish nationwide administrative registers.[25] Overall, 5964 patients with a hospital-confirmed diagnosis of HS were matched with 29 404 controls. The adjusted incidence rate ratio (IRR) was 1·57 (95% CI 1·14–2·17) for myocardial infarction and 1·33 (95% CI 1·01–1·76) for ischaemic stroke. The adjusted IRR for CVD-associated mortality was 1·95 (95% CI 1·42–2·67). When patients with severe psoriasis were used as the control group, the adjusted IRR for CVD-associated mortality remained significantly higher in the HS cohort (IRR 1·58, 95% CI 1·17–2·12). Death from CVD is the largest component of higher all-cause mortality in HS. A Finnish registry study found mean age at death was 60·5 years in people with HS, compared with 71·1 years in psoriasis and 75·2 years in naevi controls.[26]

Chronic inflammatory diseases

In European and North American populations, case–control studies demonstrate a strong association between HS and

Table 3 Hidradenitis suppurativa disease associations from case–control studies[a]

Reference (first author, year)	Odds ratio (95% CI)											
	Pilonidal sinus	Acne vulgaris	Crohn disease	Ulcerative colitis	Type 2 diabetes	Metabolic syndrome	Depression	Inflammatory arthritis	PCOS	Psoriasis
Sabat 2012[23]					4·09 (1·59–10·84)	4·46 (2·02–9·96)				
Shlyankevich 2014[37]					1·72 (1·00–2·96)				13·7 (4·00–47·3)	
Shalom 2015[60]					1·41 (1·19–1·66)	1·61 (1·36–1·89)				
Shavit 2015[62]							1·7 (1·4–2·1)			
Egeberg 2017[29]			2·04 (1·59–2·62)	1·75 (1·44–2·13)						
Ingram 2018[8]	5·61 (4·41–7·12)	1·77 (1·66–1·88)	2·65 (1·89–3·72)	1·24 (0·79–1·94)	3·39 (3·09–3·71)		1·69 (1·62–1·77)	1·06 (0·87–1·30)	1·19 (0·96–1·49)	1·09 (0·91–1·31)
Lee 2018[11]	4·97 (4·34–5·69)		1·23 (0·92–1·63)	1·32 (1·04–1·70)	1·82 (1·73–1·92)			AS 1·47 (1·14–1·89)		4·58 (4·21–4·97)

[a]In all studies, control participants are from the general population. AS, ankylosing spondylitis; CI, confidence interval; PCOS, polycystic ovary syndrome.

Crohn disease, independent of their mutual association with smoking. Prevalence of Crohn disease was 2% in patients with HS in the USA, compared with 0·6% in controls, and multivariable analysis demonstrated that patients with HS had 3·05 (95% CI 2·87–3·25) times the odds of having Crohn disease compared with those without HS.[27] The prevalence of Crohn disease in European patients was even higher at 2·5%.[28] However, there was no association between HS and Crohn disease in Korean patients.[11]

Data from four studies conducted in Denmark,[29] the UK,[8] Israel[30] and South Korea[11] were included in a meta-analysis investigating the occurrence of ulcerative colitis in patients with HS.[31] In this case there was an association between HS and ulcerative colitis in Korean patients and the pooled OR was 1·51 (95% CI 1·25–1·82).

Data regarding any association between HS and psoriasis are somewhat conflicting. A study from the UK found no association,[8] whereas studies from South Korea and Denmark found strong associations with ORs of 4·58 (95% CI 4·21–4·97) and 2·99 (95% CI 2·04–4·38), respectively.[11,32] It should be noted that biological therapy is associated with paradoxical development of HS in a few patients with psoriasis and paradoxical psoriasis in a minority of patients with HS.[33]

In South Korean patients, an association was demonstrated between HS and ankylosing spondylitis (OR 1·47, 95% CI 1·14–1·89) and rheumatoid arthritis (OR 1·31, 95% CI 1·16–1·47).[11] Among commercially insured patients in the USA, longitudinal claims data showed that patients with HS had an increased risk of developing ankylosing spondylitis compared with those without HS [hazard ratio (HR) 1·65, 95% CI 1·15–2·35].[34] There were also modest but significant associations with development of psoriatic arthritis (HR 1·44, 95% CI 1·08–1·93) and rheumatoid arthritis (HR 1·16, 95% CI 1·03–1·31).

Mental health

In keeping with HS being a socially isolating, painful chronic condition, a systematic review and meta-analysis revealed a high prevalence of depression, 21%, in those with HS.[35] An analysis of nine case–control studies gave an OR for the association between HS and depression of 1·99 (95% CI 1·63–2·43). There was a similar association between HS and anxiety, with a prevalence of 12% and a pooled OR of 1·97 (95% CI 1·65–2·35).

Perhaps in part secondary to increased mental health problems, a higher risk of completed suicide was found in a retrospective cohort study using Danish national registry data adjusted for confounding factors.[36] The HR for completed suicide in patients with HS compared with those without HS was 2·42 (95% CI 1·07–5·45).

Other medical conditions

There is considerable overlap between the demographics of HS and polycystic ovary syndrome (PCOS) and anti-androgen therapy can be helpful in both conditions. Is there an association between the two conditions? Nearly all case–control studies confirm an association, with a very high OR of 13·7 (95% CI 4·00–47·3) in one study from the USA[37] and an OR of 2·14 (95% CI 2·04–2·24) in another, which found a prevalence of 9·0% for PCOS in those with HS.[38] A study from the UK did not find an association. While there was a slightly higher prevalence of PCOS in people with HS compared with controls, statistical significance was not reached (OR 1·19, 95% CI 0·96–1·49).[8]

An association between HS and Down syndrome was demonstrated by a cross-sectional analysis of electronic medical records of 48 million individuals in the USA.[39] The adjusted odds of HS in people with Down syndrome was 5·24 (95% CI 4·62–5·94) times the odds in those without Down syndrome. The diagnosis of HS was made by the age of 29 years in 82% of people with Down syndrome compared with only 34% of people without Down syndrome. It may be that HS occurs at a younger age in those with Down syndrome or that age of onset is unaffected and instead there is earlier recognition of HS.

In the context that obesity is associated with both HS and obstructive sleep apnoea (OSA), it is intuitively likely that HS is linked to OSA. An association was confirmed in routinely collected healthcare data with an OSA prevalence of 3·5% in those with HS and an OR compared with controls of 1·45 (95% CI 1·33–1·57).[40]

Transformation of the chronic inflammation of HS into cutaneous squamous cell carcinoma (cSCC) is rare and so the literature is limited to case reports and small case series. A review of the literature in 2016 identified a total of 80 cases.[41] Most cases were in men (87%) and the average age at diagnosis was 52 years. Nearly all patients (95%) had involvement of the perianal, perineal or buttock regions. Patients often presented late, with 55% having metastases at initial presentation, probably due to a combination of cSCC being difficult to distinguish clinically from HS and the potential for contiguous spread via HS skin tunnels.

Pyoderma gangrenosum (PG) shares some of the clinical and epidemiological features of HS and both conditions are components of several autoinflammatory syndromes. Multivariable analysis of routinely collected USA healthcare data demonstrated that the odds of someone with HS having concomitant PG were 21·1 (95% CI 17·5–25·5) times the odds in the absence of HS.[42] The prevalence of PG was substantially higher in those with HS and Crohn disease compared with those with HS alone, 3·68% compared with 0·12%, respectively. When patients with Crohn disease were excluded from the analysis, those with HS were still much more likely to have PG than those without HS (OR 26·5, 95% CI 21·1–33·4), which suggests that the association between HS and PG is independent of Crohn disease status.

Some of the association between HS and PG is accounted for by the autoinflammatory syndromes PASH (PG, acne conglobata and HS), PAPASH (pyogenic arthritis, acne, PG and HS) and PASS (PG, acne vulgaris, HS and ankylosing

spondylitis).[43] While the epidemiology of these syndromes has not been fully described, the literature is limited to case reports and small case series and so the syndromes are probably relatively rare and their contribution to the overall association between HS and PG is likely to be small.

A meta-analysis of case–control studies investigating thyroid disorders in patients with HS demonstrated an OR of 1·88 (95% CI 1·25–2·81) in favour of an association.[44] After adjustment for age, sex, BMI and oral contraception in one of the case–control studies, the associations between HS and subclinical hyperthyroidism and clinical hyperthyroidism remained significant, whereas there was no association with hypothyroidism.[45] The clinical significance of the association between HS and hyperthyroidism is attenuated by relatively low absolute increases in prevalence.

Renal amyloidosis is a potential issue secondary to the inflammatory load of HS. Renal function was compared between Danish control individuals, 32 patients with HS in secondary care and 430 people with HS identified by responses to the Danish General Suburban Population Study (GESUS) questionnaire.[46] After adjusting for confounders, there was no difference in estimated glomerular filtration rate (eGFR) between individuals with HS in the population and controls but there was a higher eGFR in patients with HS in secondary care compared with controls, the mean difference being 6·81 mL min^{-1} 1·73 m^{-2} (95% CI 1·27–12·35 mL min^{-1} 1·73 m^{-2}). The results suggest that while most people with HS do not have renal dysfunction, there may be an association between more severe HS and a degree of glomerular hyperfiltration, a marker of potential renal dysfunction.

In a parallel study to the investigation of eGFR, haemoglobin (Hb) levels were examined to look for any association between HS and anaemia, in the context that people with HS frequently report fatigue. Unadjusted and adjusted analysis demonstrated no difference in Hb levels or rates of anaemia between Danish controls and individuals with HS in the population or the hospital HS group.[47] In contrast, a case–control study of 1431 patients with HS in the USA compared with a control group of 8518 patients with acne vulgaris did find an association between HS and anaemia.[48] The OR for anaemia in patients with HS compared with patients with acne after controlling for BMI and HbA_{1C} was 2·20 (95% CI 1·42–3·41). The association was particularly strong in male patients with an OR of 5·61 (95% CI 1·86–16·90). There was a dose–response effect observed in males, with the odds of moderate/severe anaemia, defined as Hb equal to or less than 11·0 g dL^{-1}, being 9·07 (95% CI 1·98–41·43) times higher in patients with HS compared with patients with acne.

Discovery that loss-of-function mutations in γ-secretase genes cause HS in some Han Chinese patients[49] led to a search for a possible association between HS and Alzheimer disease, on the basis that familial Alzheimer disease is caused by γ-secretase missense mutations.[50] However, there is no current support for a link between HS and Alzheimer disease, with several case–control studies finding no association.[8,51,52]

Hepatic ultrasonography was used to show a 72·9% prevalence of nonalcoholic fatty liver disease in patients with HS compared with 24·7% in age- and sex-matched controls and after adjustment for classic metabolic risk factors, the OR was 7·75 (CI 2·5–23·6).[53]

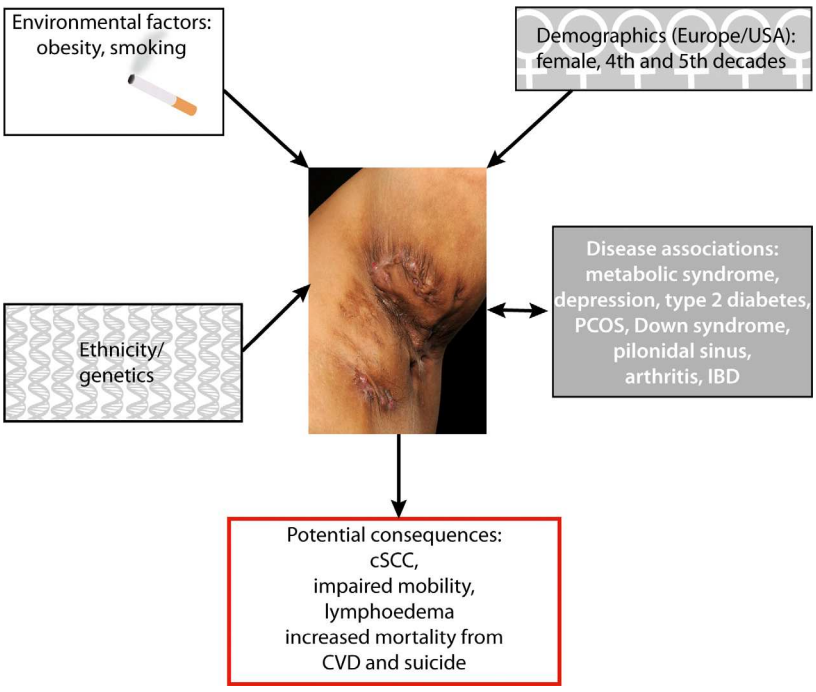

Figure 1 Summary of the epidemiology of hidradenitis suppurativa (HS). CVD, cardiovascular disease; IBD, inflammatory bowel disease; PCOS, polycystic ovary syndrome; cSCC, cutaneous squamous cell carcinoma.

Hidradenitis suppurativa phenotypic subtypes

Our understanding of the epidemiology of HS is likely to evolve as we begin to recognize HS phenotypic subtypes. Differences are already apparent in the male-to-female ratio for HS in European compared with Asian populations. There have been several attempts to identify HS subphenotypes, including latent class analysis which divided patients into three classes: (i) predominantly axillary and chest involvement mainly affecting women; (ii) a follicular subtype associated with severe acne mainly affecting men; and (iii) a gluteal form with predominantly buttock involvement.[54,55]

Natural history

Investigation of the natural history of HS is limited by a relative lack of prospective cohort studies. A retrospective analysis of 846 patients with HS from the Netherlands identified five severity risk factors, namely male sex, obesity, smoking pack-years, disease duration and lesions in the axillary, perianal and mammary regions.[19] Positive family history was linked to earlier disease onset but not to more severe disease.

A cross-sectional postal questionnaire study assessed risk factors, clinical course and prognosis in 141 Danish patients with HS and 79 Dutch patients with HS under follow-up for a median of 22 years.[56] From the 129 fully completed responses, rates of remission, improvement, unchanged severity and worsening were 39%, 32%, 21% and 8%, respectively. Current nonsmokers were more likely to report remission than active smokers (40% vs. 29%) and a higher proportion of nonobese patients (45%) reported remission compared with obese patients (23%).

A further cross-sectional survey of female patients with HS in the Netherlands investigated the influence of menses and pregnancy on disease severity.[57] Of the 186 respondents, 43% reported a deterioration of HS at the time of menses, while 54% reported no change. Pregnancy was associated with HS improvement, no change, or worsening in 30%, 53% and 17%, respectively. Improvement during pregnancy correlated with worsening at the time of menses.

Disease onset at or soon after puberty is supported by the peak in new HS diagnoses occurring in those aged 18–29 years, factoring in an average delay in diagnosis of 7 years.[2,14] It has been suggested that there could be correspondingly higher rates of remission in women reaching menopause. In support of this theory, the female-to-male ratio alters from 5 : 1 for the age range 20–29 years to 3 : 1 for those aged 50–59 years.[8] However, prospective data are not yet available.

Conclusion

Our understanding of the epidemiology of HS has greatly increased in the last decade. The use of routinely collected healthcare data has permitted population-based studies of prevalence and incidence and case–control studies have established multiple associations between HS and other medical conditions, in particular CVD, inflammatory bowel disease and depression (Figure 1). Mortality rates from CVD are more than 50% higher in HS than in psoriasis and there is an increased risk of completed suicide compared with controls. Smoking and obesity are closely associated with HS and may also increase disease severity.

Several unanswered questions remain. What is the true prevalence of HS in Europe and North America, when prevalence figures vary by an order of magnitude? What is the natural history of HS, particularly regarding predictors of disease trajectory? In addition, is the epidemiology of HS uniform across different phenotypic subclasses and across populations with differing ethnicity? There is still a lot of work to be done.

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